> d 16 1-15, ab

L6 ANSWER 1 OF 15 MEDLINE
AB BACKGROUND: Translation initiation factor 4A (eIF4A) is the prototype of the **DEAD-box** family of proteins. **DEAD-box** proteins are involved in a variety of cellular processes including splicing, ribosome biogenesis and **RNA** degradation. Energy from ATP hydrolysis is used to perform **RNA** unwinding during initiation of mRNA translation. The presence of eIF4A is required for the 43S preinitiation complex to bind to and scan the mRNA. RESULTS: We present here the crystal structure of the nucleotide-binding domain of eIF4A at 2.0 A and the structures with bound adenosinediphosphate and adenosinetriphosphate at 2.2 A and 2.4 A resolution, respectively. The structure of the apo form of the enzyme has been determined by multiple isomorphous replacement. The ATPase domain contains a central seven-stranded beta sheet flanked by nine alpha helices. Despite low sequence homology to the NTPase domains of **RNA** and DNA helicases, the three-dimensional fold of eIF4A is nearly identical to the DNA **helicase** PcrA of Bacillus stearothermophilus and to the **RNA helicase** NS3 of **hepatitis C** virus. CONCLUSIONS: We have determined the crystal structure of the N-terminal domain of the eIF4A from yeast as the first structure of a member of the **DEAD-box** protein family. The complex of the protein with bound ADP and ATP offers insight into the mechanism of ATP hydrolysis and the transfer of energy to unwind **RNA**. The identical fold of the ATPase domain of the DNA **helicase** PcrA of B. stearothermophilus and the **RNA helicase** of **hepatitis C** virus suggests a common fold for all ATPase domains of DExx- and **DEAD-box** proteins.

L6 ANSWER 2 OF 15 MEDLINE
AB Approximately 4 million Americans are infected with the **hepatitis C** virus (HCV), making it a major cause of chronic liver disease. Because of the lack of an efficient cell culture system, little is known about the interaction between HCV and host cells. We performed a yeast two-hybrid screen of a human liver cell cDNA library with HCV core protein as bait and isolated the **DEAD box** protein DBX. DBX has significant amino acid sequence identity to mouse PL10, an ATP-dependent **RNA helicase**. The binding of DBX to HCV core protein occurred in an in vitro binding assay in the presence of 1 M NaCl or detergent. When expressed in mammalian cells, HCV core protein and DBX were co-localized at the endoplasmic reticulum. In a mutant strain of Saccharomyces cerevisiae, DBX complemented the function of Ded1p, an essential **DEAD box RNA helicase**. HCV core protein inhibited the growth of DBX-complemented mutant yeast but not Ded1p-expressing yeast. HCV core protein also inhibited the in vitro translation of capped but not uncapped **RNA**. These findings demonstrate an interaction between HCV core protein and a host cell protein involved in **RNA** translation and suggest a mechanism by which HCV may inhibit host cell mRNA translation.

L6 ANSWER 3 OF 15 MEDLINE
AB Several studies have implicated **hepatitis C** virus (HCV) core in influencing the expression of host genes. To identify cellular factors with a possible role in HCV replication and pathogenesis,

we looked for cellular proteins that interact with the viral core protein.
A human liver cDNA library was screened in a yeast two-hybrid assay to identify cellular proteins that bind to core. Several positive clones were isolated, one of which encoded the C-terminal 253 amino acids of a putative **RNA helicase**, a **DEAD box** protein designated DDX3. Bacterially expressed glutathione-S-transferase-DDX3 fusion protein specifically pulled down in vitro translated and radiolabeled HCV core, confirming a direct interaction. Immunofluorescent staining of HeLa cells with a polyclonal antiserum showed that DDX3 is located predominantly in nuclear speckles and at low levels throughout the cytoplasm. In cells infected with a recombinant vaccinia virus expressing HCV structural proteins (core, E1, and E2), DDX3 and core colocalized in distinct spots in the perinuclear region of the cytoplasm. The regions of the proteins involved in binding were found by deletion analysis to be the N-terminal 59 amino acid residues of core and a C-terminal RS-like domain of DDX3. The human DDX3 is a putative **RNA helicase** and a member of a highly conserved **DEAD box** subclass that includes murine PL10, Xenopus An3, and yeast Ded1 proteins. Their role in **RNA** metabolism or gene expression is unknown. The significance of core-**helicase** interaction in HCV replication and pathogenesis is discussed. Copyright 1999 Academic Press.

L6 ANSWER 4 OF 15 MEDLINE

AB The nucleocapsid core protein of **hepatitis C** virus (HCV) has been shown to trans-act on several viral or cellular promoters. To get insight into the trans-action mechanism of HCV core protein, a yeast two-hybrid cloning system was used for identification of core protein-interacting cellular protein. One such cDNA clone encoding the **DEAD box** family of putative **RNA helicase** was obtained. This cellular putative **RNA helicase**, designated CAP-Rf, exhibits more than 95% amino acid sequence identity to other known **RNA** helicases including human DBX and DBY, mouse mDEAD3, and PL10, a family of proteins generally involved in translation, splicing, development, or cell growth. In vitro binding or in vivo coimmunoprecipitation studies demonstrated the direct interaction of the full-length/matured form and C-terminally truncated variants of HCV core protein with this targeted protein. Additionally, the protein's interaction domains were delineated at the N-terminal 40-amino-acid segment of the HCV core protein and the C-terminal tail of CAP-Rf, which encompassed its **RNA**-binding and ATP hydrolysis domains. Immunoblotting or indirect immunofluorescence analysis revealed that the endogenous CAP-Rf was mainly localized in the nucleus and to a lesser extent in the cytoplasm, and when fused with FLAG tag, it colocalized with the HCV core protein either in the cytoplasm or in the nucleus. Similar to other **RNA** helicases, this cellular **RNA helicase** has nucleoside triphosphatase-deoxynucleoside triphosphatase activity, but this activity is inhibited by various forms of homopolynucleotides and enhanced by the HCV core protein.
Moreover, transient expression of HCV core protein in human hepatoma HuH-7 cells significantly potentiated the trans-activation effect of FLAG-tagged CAP-Rf or untagged CAP-Rf on the luciferase reporter plasmid activity. All together, our results indicate that CAP-Rf is involved in regulation of gene expression and that HCV core protein promotes the trans-activation ability of CAP-Rf, likely via the complex formation and the modulation of the ATPase-dATPase activity of CAP-Rf. These findings provide evidence

that HCV may have evolved a distinct mechanism in alteration of host cellular gene expression regulation via the interaction of its nucleocapsid core protein and cellular putative **RNA helicase** known to participate in all aspects of cellular processes involving **RNA** metabolism. This feature of core protein may impart pleiotropic effects on host cells, which may partially account for its role in HCV pathogenesis.

L6 ANSWER 5 OF 15 MEDLINE

AB The carboxyl-terminal three-fourths of the **hepatitis C** virus (HCV) NS3 protein has been shown to possess an **RNA helicase** activity, typical of members of the **DEAD box** family of **RNA** helicases. In addition, the NS3 protein contains four amino acid motifs conserved in **DEAD box** proteins. In order to inspect the roles of individual amino acid residues in the four conserved motifs (AXXXXGKS, DECH, TAT, and QRRGRTGR) of the NS3 protein, mutational analysis was used in this study. Thirteen mutant proteins were constructed, and their biochemical activities were examined. Lys1235 in the AXXXXGKS motif was important for basal nucleoside triphosphatase (NTPase) activity in the absence of polynucleotide cofactor. A serine in the X position of the DEXH motif disrupted the NTPase and **RNA helicase** activities. Alanine substitution at His1318 of the DEXH motif made the protein possess high NTPase activity. In addition, we now report inhibition of NTPase activity of NS3 by polynucleotide cofactor. Gln1486 was indispensable for the enzyme activity, and this residue represents a distinguishing feature between **DEAD box** and DEXH proteins. There are four Arg residues in the QRRGRTGR motif of the HCV NS3 protein, and the second, Arg1488, was important for **RNA** binding and enzyme activity, even though it is less well conserved than other Arg residues. Arg1490 and Arg1493 were essential for the enzymatic activity. As the various enzymatic activities were altered by mutation, the enzyme characteristics were also changed.

L6 ANSWER 6 OF 15 MEDLINE

AB The NS3 protein of **hepatitis C** virus contains a bipartite structure consisting of an N-terminal serine protease and a C-terminal **DEAD box helicase**. We show that the C-terminal domain has ATPase and panhelicase activities. The integrity of the **helicase** function is dependent on the conserved **DEAD** motif and can be abolished by a His-Ala point mutation, leaving a fully functional nucleoside triphosphatase.

L6 ANSWER 7 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS

AB Background: Translation initiation factor 4A (eIF4A) is the prototype of the **DEAD-box** family of proteins. **DEAD-box** proteins are involved in a variety of cellular processes including splicing, ribosome biogenesis and **RNA** degradation. Energy from ATP hydrolysis is used to perform **RNA** unwinding during initiation of mRNA translation. The presence of eIF4A is required for the 43S preinitiation complex to bind to and scan the mRNA. Results: We present here the crystal structure of the nucleotide-binding domain of eIF4A at 2.0 ANG and the structures with bound adenosinediphosphate and adenosinetriphosphate at 2.2 ANG and 2.4 ANG resolution, respectively. The structure of the apo form of the enzyme has been determined by multiple isomorphous replacement. The ATPase domain contains a central seven-stranded beta sheet flanked by nine alpha helices. Despite low sequence homology to the NTPase domains of **RNA** and DNA helicases, the three-dimensional fold of eIF4A is nearly identical to the DNA **helicase** PcrA of Bacillus stearothermophilus and to the **RNA helicase** NS3 of **hepatitis C** virus. Conclusions: We have determined the crystal structure of the

N-terminal domain of the eIF4A from yeast as the first structure of a member of the **DEAD-box** protein family. The complex of the protein with bound ADP and ATP offers insight into the mechanism of ATP hydrolysis and the transfer of energy to unwind **RNA**. The identical fold of the ATPase domain of the DNA **helicase** PcrA of B. stearothermophilus and the **RNA helicase** of **hepatitis C** virus suggests a common fold for all ATPase domains of DExx- and **DEAD-box** proteins.

L6 ANSWER 8 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS
AB Approximately 4 million Americans are infected with the **hepatitis C** virus (HCV), making it a major cause of chronic liver disease. Because of the lack of an efficient cell culture system, little is known about the interaction between HCV and host cells. We performed a yeast two-hybrid screen of a human liver cell cDNA library with HCV core protein as bait and isolated the **DEAD box** protein DBX. DBX has significant amino acid sequence identity to mouse PL10, an ATP-dependent **RNA helicase**. The binding of DBX to HCV core protein occurred in an in vitro binding assay in the presence of 1 M NaCl or detergent. When expressed in mammalian cells, HCV core protein and DBX were co-localized at the endoplasmic reticulum. In a mutant strain of Saccharomyces cerevisiae, DBX complemented the function of Ded1p, an essential **DEAD box RNA helicase**. HCV core protein inhibited the growth of DBX-complemented mutant yeast but not Ded1p-expressing yeast. HCV core protein also inhibited the in vitro translation of capped but not uncapped **RNA**. These findings demonstrate an interaction between HCV core protein and a host cell protein involved in **RNA** translation and suggest a mechanism by which HCV may inhibit host cell mRNA translation.

L6 ANSWER 9 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS
AB Several studies have implicated **hepatitis C** virus (HCV) core in influencing the expression of host genes. To identify cellular factors with a possible role in HCV replication and pathogenesis, we looked for cellular proteins that interact with the viral core protein. A human liver cDNA library was screened in a yeast two-hybrid assay to identify cellular proteins that bind to core. Several positive clones were isolated, one of which encoded the C-terminal 253 amino acids of a putative **RNA helicase**, a **DEAD box** protein designated DDX3. Bacterially expressed glutathione-S-transferase-DDX3 fusion protein specifically pulled down in vitro translated and radiolabeled HCV core, confirming a direct interaction. Immunofluorescent staining of HeLa cells with a polyclonal antiserum showed that DDX3 is located predominantly in nuclear speckles and at low levels throughout the cytoplasm. In cells infected with a recombinant vaccinia virus expressing HCV structural proteins (core, E1, and E2), DDX3and core colocalized in distinct spots in the perinuclear region of the cytoplasm. The regions of the proteins involved in binding were found by deletion analysis to be the N-terminal 59 amino acid residues of core and a C-terminal RS-like domain of DDX3. The human DDX3 is a putative **RNA helicase** and a member of a highly conserved **DEAD box** subclass that includes murine PL10, Xenopus An3, and yeast Ded1 proteins. Their role in **RNA** metabolism or gene expression is unknown. The significance of core-**helicase** interaction in HCV replication and pathogenesis is discussed.

L6 ANSWER 10 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS
AB The nucleocapsid core protein of **hepatitis C** virus

(HCV) has been shown to trans-act on several viral or cellular promoters. To get insight into the trans-action mechanism of HCV core protein, a yeast two-hybrid cloning system was used for identification of core protein-interacting cellular protein. One such cDNA clone encoding the **DEAD box** family of putative **RNA helicase** was obtained. This cellular putative **RNA helicase**, designated CAP-Rf, exhibits more than 95% amino acid sequence identity to other known **RNA** helicases including human DBX and DBY, mouse mDEAD3, and PL10, a family of proteins generally involved in translation, splicing, development, or cell growth. In vitro binding or in vivo coimmunoprecipitation studies demonstrated the direct interaction of the full-length/matured form and C-terminally truncated variants of HCV core protein with this targeted protein. Additionally, the protein's interaction domains were delineated at the N-terminal 40-amino-acid segment of the HCV core protein and the C-terminal tail of CAP-Rf, which encompassed its **RNA**-binding and ATP hydrolysis domains. Immunoblotting or indirect immunofluorescence analysis revealed that the endogenous CAP-Rf was mainly localized in the nucleus and to a lesser extent in the cytoplasm, and when fused with FLAG tag, it colocalized with the HCV core protein either in the cytoplasm or in the nucleus. Similar to other **RNA** helicases, this cellular **RNA helicase** has nucleoside triphosphatase-deoxynucleoside triphosphatase activity, but this activity is inhibited by various forms of homopolynucleotides and enhanced by the HCV core protein. Moreover, transient expression of HCV core protein in human hepatoma HuH-7 cells significantly potentiated the trans-activation effect of FLAG-tagged CAP-Rf or untagged CAP-Rf on the luciferase reporter plasmid activity. All together, our results indicate that CAP-Rf is involved in regulation of gene expression and that HCV core protein promotes the trans-activation ability of CAP-Rf, likely via the complex formation and the modulation of the ATPase-dATPase activity of CAP-Rf. These findings provide evidence that HCV may have evolved a distinct mechanism in alteration of host cellular gene expression regulation via the interaction of its nucleocapsid core protein and cellular putative **RNA helicase** known to participate in all aspects of cellular processes involving **RNA** metabolism. This feature of core protein may impart pleiotropic effects on host cells, which may partially account for its role in HCV pathogenesis.

L6 ANSWER 11 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS

L6 ANSWER 12 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS

AB The carboxyl-terminal three-fourths of the **hepatitis C** virus (HCV) NS3 protein has been shown to possess an **RNA helicase** activity, typical of members of the **DEAD box** family of **RNA** helicases. In addition, the NS3 protein contains four amino acid motifs conserved in **DEAD box** proteins. In order to inspect the roles of individual amino acid residues in the four conserved motifs (AXXXXGKS, DECH, TAT, and QRRGRTGR) of the NS3 protein, mutational analysis was used in this study. Thirteen mutant proteins were constructed, and their biochemical activities were examined. Lys1235 in the AXXXXGKS motif was important for basal nucleoside triphosphatase (NTPase) activity in the absence of polynucleotide cofactor. A serine in the X position of the DEXH motif disrupted the NTPase and **RNA helicase** activities. Alanine substitution at His1318 of the DEXH motif made the protein possess high NTPase activity. In addition, we now report inhibition of NTPase activity of NS3 by polynucleotide cofactor. Gln1486 was indispensable for

the enzyme activity, and this residue represents a distinguishing feature between **DEAD box** and DEXH proteins. There are four Arg residues in the QRRGRTGR motif of the HCV NS3 protein, and the second, Arg1488, was important for **RNA** binding and enzyme activity, even though it is less well conserved than other Arg residues. Arg1490 and Arg1493 were essential for the enzymatic activity. As the various enzymatic activities were altered by mutation, the enzyme characteristics were also changed.

L6 ANSWER 13 OF 15 USPATFULL

AB The invention relates to the X-ray crystal structure of the **hepatitis C** virus **helicase** domain. More specifically, the invention relates to crystallized complexes of HCV **helicase** and an oligonucleotide, to crystallizable compositions of HCV **helicase** and an oligonucleotide and to methods of crystallizing an HCV **helicase**-oligonucleotide complex. The invention further relates to a computer programmed with the structure coordinates of the HCV **helicase** oligonucleotide binding pocket or the HCV **helicase** nucleotide triphosphate pocket wherein said computer is capable of displaying a three-dimensional representation of that binding pocket.

L6 ANSWER 14 OF 15 USPATFULL

AB The present invention relates to the determination of an authentic HCV genome **RNA** sequences, to construction of infectious HCV DNA clones, and to use of the clones, or their derivatives, in therapeutic, vaccine, and diagnostic applications. The invention is also directed to HCV vectors, e.g., for gene therapy of gene vaccines.

L6 ANSWER 15 OF 15 USPATFULL

AB The **Hepatitis C** Virus (HCV) NS3 protein contains amino acid motifs of a serine proteinase, a nucleotide triphosphatase (NTPase), and an **RNA helicase**. A carboxy fragment of the HCV NS3 protein was purified and possessed **RNA helicase** activity. Detections from the amino terminus resulted in the protein becoming soluble. Deletions from the carboxy terminus do not result in a loss of **helicase** activity until at least 50 amino acids are deleted. The **helicase** activity requires ATP and divalent cations such as Mg.sup.2+ and Mn.sup.2+. The **helicase** activity was blocked by monoclonal antibody specific to the HCV NS3 protein.

=> d 16 1-15

L6 ANSWER 1 OF 15 MEDLINE

AN 1999332675 MEDLINE

DN 99332675

TI Crystal structure of the ATPase domain of translation initiation factor 4A from Saccharomyces cerevisiae--the prototype of the **DEAD box** protein family.

AU Benz J; Trachsel H; Baumann U

CS Departement fur Chemie und Biochemie, Universitat Bern, Germany.. joerg.benz@ibc.unibe.ch

SO Structure Fold Des, (1999 Jun 15) 7 (6) 671-9. Journal code: DEB.

CY ENGLAND: United Kingdom

DT Journal; Article; (JOURNAL ARTICLE)

LA English

FS Priority Journals

EM 199911

EW 19991101

L6 ANSWER 2 OF 15 MEDLINE
AN 1999269118 MEDLINE
DN 99269118
TI **Hepatitis C** virus core protein binds to a **DEAD box RNA helicase.**
AU Mamiya N; Worman H J
CS Departments of Medicine and of Anatomy and Cell Biology, College of Physicians and Surgeons, Columbia University, New York, New York 10032, USA.
NC 1S10 RR10506 (NCRR)
5 P30 CA13696 (NCI)
SO JOURNAL OF BIOLOGICAL CHEMISTRY, (1999 May 28) 274 (22) 15751-6.
Journal code: HIV. ISSN: 0021-9258.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199909
EW 19990901

L6 ANSWER 3 OF 15 MEDLINE
AN 1999263161 MEDLINE
DN 99263161
TI **Hepatitis C** virus core protein interacts with a human **DEAD box** protein DDX3.
AU Owsianka A M; Patel A H
CS Medical Research Council Virology Unit, Church Street, Glasgow, G11 5JR, United Kingdom.
SO VIROLOGY, (1999 May 10) 257 (2) 330-40.
Journal code: XEA. ISSN: 0042-6822.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199908
EW 19990803

L6 ANSWER 4 OF 15 MEDLINE
AN 1999173979 MEDLINE
DN 99173979
TI **Hepatitis C** virus core protein interacts with cellular putative **RNA helicase.**
AU You L R; Chen C M; Yeh T S; Tsai T Y; Mai R T; Lin C H; Lee Y H
CS Institute of Biochemistry, National Yang-Ming University, Taipei, Taiwan 112, Republic of China.
SO JOURNAL OF VIROLOGY, (1999 Apr) 73 (4) 2841-53.
Journal code: KCV. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199907
EW 19990702

L6 ANSWER 5 OF 15 MEDLINE
AN 1998037651 MEDLINE
DN 98037651
TI Mutational analysis of the **hepatitis C** virus **RNA helicase.**
AU Kim D W; Kim J; Gwack Y; Han J H; Choe J
CS Department of Biological Sciences, Korea Advanced Institute of Science and Technology, Taejon.
SO JOURNAL OF VIROLOGY, (1997 Dec) 71 (12) 9400-9.

Journal code: KCV. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199803

L6 ANSWER 6 OF 15 MEDLINE
AN 97366700 MEDLINE
DN 97366700
TI A point mutation abolishes the **helicase** but not the nucleoside triphosphatase activity of **hepatitis C** virus NS3 protein.
AU Heilek G M; Peterson M G
CS Tularik Inc., South San Francisco, California 94080, USA.
SO JOURNAL OF VIROLOGY, (1997 Aug) 71 (8) 6264-6.
Journal code: KCV. ISSN: 0022-538X.
CY United States
DT Journal; Article; (JOURNAL ARTICLE)
LA English
FS Priority Journals; Cancer Journals
EM 199710

L6 ANSWER 7 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1999:341537 BIOSIS
DN PREV199900341537
TI Crystal structure of the ATPase domain of translation initiation factor 4A
from Saccharomyces cerevisiae - The prototype of the **DEAD box** protein family.
AU Benz, Joerg (1); Trachsel, Hans; Baumann, Ulrich (1)
CS (1) Departement fuer Chemie und Biochemie, Universitaet Bern, Freiestrasse
3, Bern Germany
SO Structure (London), (June, 1999) Vol. 7, No. 6, pp. 671-679.
ISSN: 0969-2126.
DT Article
LA English
SL English

L6 ANSWER 8 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1999:334112 BIOSIS
DN PREV199900334112
TI **Hepatitis C** virus core protein binds to a **DEAD box RNA helicase.**
AU Mamiya, Naoto; Worman, Howard J. (1)
CS (1) Dept. of Medicine, College of Physicians and Surgeons, Columbia University, 630 West 168th St., 10th Floor, Rm. 508, New York, NY, 10032 USA
SO Journal of Biological Chemistry, (May 28, 1999) Vol. 274, No. 22, pp. 15751-15756.
ISSN: 0021-9258.
DT Article
LA English
SL English

L6 ANSWER 9 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1999:273702 BIOSIS
DN PREV199900273702
TI **Hepatitis C** virus core protein interacts with a human **DEAD box** protein DDX3.
AU Owsianka, Ania M.; Patel, Arvind H. (1)
CS (1) Medical Research Council Virology Unit, Church Street, Glasgow, G11 5JR UK
SO Virology, (May 10, 1999) Vol. 257, No. 2, pp. 330-340.

ISSN: 0042-6822.
DT Article
LA English
SL English

L6 ANSWER 10 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1999:210830 BIOSIS
DN PREV199900210830
TI **Hepatitis C** virus core protein interacts with cellular putative **RNA helicase**.
AU You, Li-Ru; Chen, Chun-Ming; Yeh, Tien-Shun; Tsai, Tzung-Yuan; Mai, Ru-Tsun; Lin, Chi-Hung; Lee, Yan-Hwa Wu (1)
CS (1) Institute of Biochemistry, National Yang-Ming University, Taipei, 112 Taiwan
SO Journal of Virology, (April, 1999) Vol. 73, No. 4, pp. 2841-2853. ISSN: 0022-538X.
DT Article
LA English
SL English

L6 ANSWER 11 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1998:526153 BIOSIS
DN PREV199800526153
TI Inhibition of a cellular **dead-box RNA helicase** by **hepatitis C** virus core protein.
AU Mamiya, N.; Woman, H. J.
CS Dep. Med., Coll. Physicians Surgeons, Columbia Univ., New York, NY USA
SO Hepatology, (Oct., 1998) Vol. 28, No. 4 PART 2, pp. 462A. Meeting Info.: Biennial Scientific Meeting of the International Association for the Study of the Liver and the 49th Annual Meeting and Postgraduate Courses of the American Association for the Study of Liver Diseases Chicago, Illinois, USA November 4-10, 1998 International Association for the Study of the Liver . ISSN: 0270-9139.
DT Conference
LA English

L6 ANSWER 12 OF 15 BIOSIS COPYRIGHT 2001 BIOSIS
AN 1998:31375 BIOSIS
DN PREV199800031375
TI Mutational analysis of the **hepatitis C** virus **RNA helicase**.
AU Kim, Dong Wook; Kim, Jiyun; Gwack, Yousang; Han, Jang H.; Choe, Joonho (1)
CS (1) Dep. Biol. Sci., Korea Advanced Inst. Sci. Technol., Taejon 305-701 South Korea
SO Journal of Virology, (Dec., 1997) Vol. 71, No. 12, pp. 9400-9409. ISSN: 0022-538X.
DT Article
LA English

L6 ANSWER 13 OF 15 USPATFULL
AN 2001:17370 USPATFULL
TI **Hepatitis C** virus **helicase** crystals and coordinates that define **helicase** binding pockets
IN Kim, Joseph L., Natick, MA, United States
Morgenstern, Kurt A., Derry, NH, United States
Caron, Paul R., Malden, MA, United States
Lin, Chao, Brookline, MA, United States
PA Vertex Pharmaceuticals Inc., Cambridge, MA, United States (U.S. corporation)
PI US 6183121 20010206
AI US 1998-128314 19980803 (9)
PRAI US 1997-55772 19970813 (60)
DT Utility

LN.CNT 2174
INCL INCLM: 364/499.000
INCLS: 364/499.000; 364/578.000; 378/071.000; 378/073.000; 378/079.000; 536/023.100; 530/364.000; 530/388.210
NCL NCLM: 364/496.000
NCLS: 364/499.000; 364/578.000; 378/071.000; 378/073.000; 378/079.000; 536/023.100; 530/364.000; 530/388.210
IC [7]
ICM: G01N023-20
ICS: C07H021-04; C07K016-00
EXF 364/499; 364/496; 364/578; 376/79; 376/71; 376/73; 536/23.1; 530/364; 530/388.21
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 14 OF 15 USPATFULL
AN 2000:131588 USPATFULL
TI Functional DNA clone for **hepatitis C** virus (HCV) and uses thereof
IN Rice, Charles M., University City, MO, United States
Kolykhalov, Alexander A., St. Louis, MO, United States
PA Washington University, St. Louis, MO, United States (U.S. corporation)
PI US 6127116 20001003
AI US 1997-811566 19970304 (8)
RLI Continuation-in-part of Ser. No. US 1995-520678, filed on 29 Aug 1995, now patented, Pat. No. US 5874565, issued on 23 Feb 1999
DT Utility
LN.CNT 5685
INCL INCLM: 435/006.000
INCLS: 435/320.100; 435/325.000; 536/023.700; 536/024.100; 536/024.300; 536/024.500
NCL NCLM: 435/006.000
NCLS: 435/320.100; 435/325.000; 536/023.700; 536/024.100; 536/024.300; 536/024.500
IC [7]
ICM: C07H021-02
ICS: C07H021-04; C12N005-10; C12N015-63
EXF 435/6; 435/69.1; 435/91.1; 435/91.3; 435/91.32; 435/91.33; 435/172.1; 435/172.3; 435/235.1; 435/236; 435/320.1; 435/370; 435/325; 435/455; 536/23.1; 536/23.72; 536/24.1; 536/24.3; 536/25.1; 935/22; 935/23; 935/24; 935/32; 935/55; 935/57; 935/66; 935/70; 935/71
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

L6 ANSWER 15 OF 15 USPATFULL
AN 1999:151008 USPATFULL
TI HCV NS3 protein fragments having **helicase** activity and improved solubility
IN Houghton, Michael, Danville, CA, United States
Choo, Qui-Lim, El Cerrito, CA, United States
Han, Jang, Lafayette, CA, United States
Choe, Joonho, Taejon, Korea, Republic of
PA Chiron Corporation, Emeryville, CA, United States (U.S. corporation)
PI US 5989905 19991123
AI US 1997-833678 19970408 (8)
RLI Division of Ser. No. US 1995-529169, filed on 15 Sep 1995
DT Utility
LN.CNT 1545
INCL INCLM: 435/320.100
INCLS: 536/023.200; 536/023.400; 536/023.720
NCL NCLM: 435/320.100
NCLS: 536/023.200; 536/023.400; 536/023.720
IC [6]
ICM: C12N015-00
ICS: C07H021-04
EXF 435/69.7; 435/320.1; 435/183; 536/23.2; 536/23.4; 536/23.72
CAS INDEXING IS AVAILABLE FOR THIS PATENT.